

Analysis of Various Responses Occur in the Cardiovascular System

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Abstract:

The cardiovascular system (also known as the circulatory system) is composed of the heart, blood, and blood vessels. The system is known as a closed system, because the blood never leaves the system of blood vessels. The cardiac output (CO) depends upon the heart rate and stroke volume. The heart rate depends on blood pressure. So, to have the normal cardiac output, using feedback through baroreflexes must regulate blood pressure. As pressure changes, heart rate also changes proportionately, and an opposite change is observed in cardiac output. Regulation of arterial blood pressure by feedback through baroreflexes is analyzed in complex dynamics. Changes in heart rate with respect to time and changes in CO with respect to blood pressure is obtained at different time delays. The response of the cardiovascular model for different values of time delay is obtained by using Simulink.

Keywords: Cardiovascular System, Cardio output, baroreflexes, blood pressure.

1. Introduction:

Cardiac Output (CO):

Cardiac Output is the amount of blood ejected from the left ventricle, (or the right ventricle) into the aorta, (or pulmonary trunk) each minute. CO is determined by an average of $CO = SV \times HR = 5.25 \text{ liters/min.}$ $CO = SV * HR.$

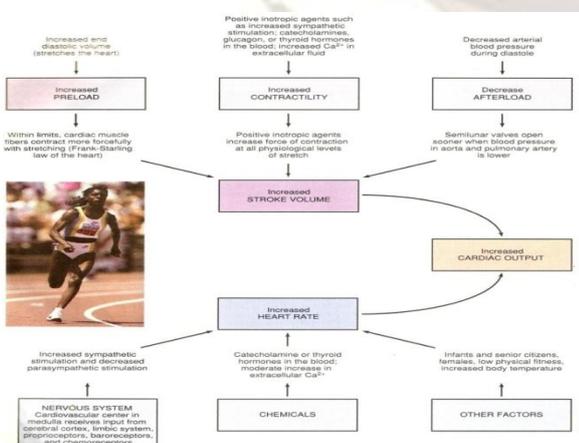


Figure 1: Factors, that increases cardiac output Blood Pressure:

Blood pressure (BP) is the pressure exerted by blood, on the wall of a blood vessel. BP is generated, by contraction of the ventricles. $BP = 120 \text{ mmHg} / 80 \text{ mmHg}.$

$$CO = MABP / R$$

$$MABP = CO * R.$$

CO and BP depends on the total volume of blood, in the cardiovascular system. Normal volume of blood in an adult is about 5 liters. If there is, any decrease in this volume, BP drops. On the other hand, anything that increases blood volume, increases BP.

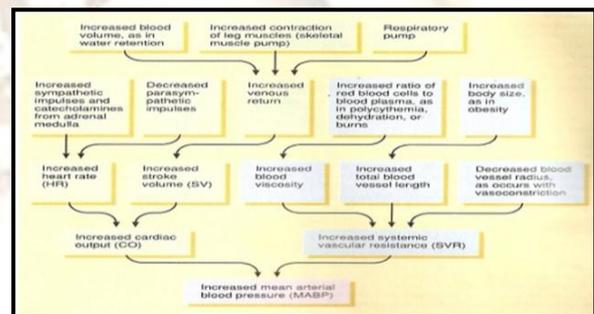


Figure 2 Summary of factors that affect blood Pressure

Control of blood pressure, and blood flow:

From moment to moment, and day to day, several inter connected negative feedback systems control blood pressure, by adjusting heart rate, stroke volume, systemic vascular resistance and blood volume.

2. Neural Regulation:

Cardio vascular Centre (CVC): CVC is the Group of neurons, within the medulla of the brain stem, regulate heart rate, contractility of the ventricles, and blood vessel diameter (vasoconstriction or vasodilatation) as shown in Figure 3. It controls BP, by slowing down or speeding up the heart rate, and by dilating or constricting blood vessels. It receives input from higher brain regions, baroreceptors and chemoreceptor. Baroreceptors are important pressure sensitive neurons, that monitors, stretching of the walls of blood vessels and the atria, and participate in several negative feedback system, that contribute to blood pressure control. Chemo

receptors monitors, blood acidity, carbon dioxide level, and oxygen level. CVC provides output to both sympathetic and parasympathetic divisions of the Autonomic nervous system (ANS). Summary of the main mechanisms in controlling blood pressure is shown in Figure 4 [2, 3]

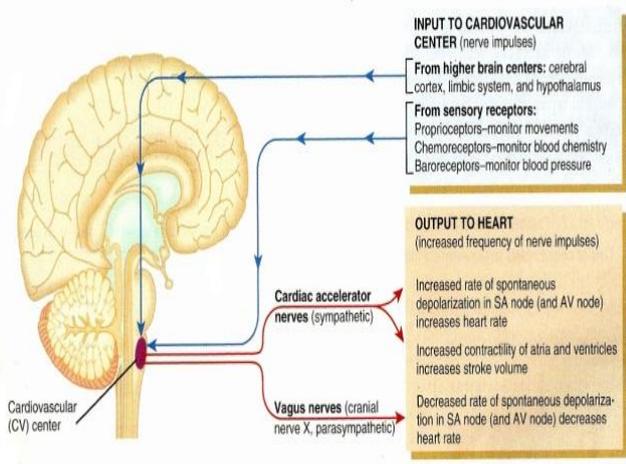


Figure 3: Cardiovascular (CV) centre

Table 1: The Sympathetic and parasympathetic nervous systems

	Sympathetic stimulation	Parasympathetic stimulation
Heart	↑ Rate ↑ Strength of Contraction	↓ Rate ↓ Strength of contraction
Blood vessels	Most Constrict	There is little parasympathetic innervations to most blood vessels

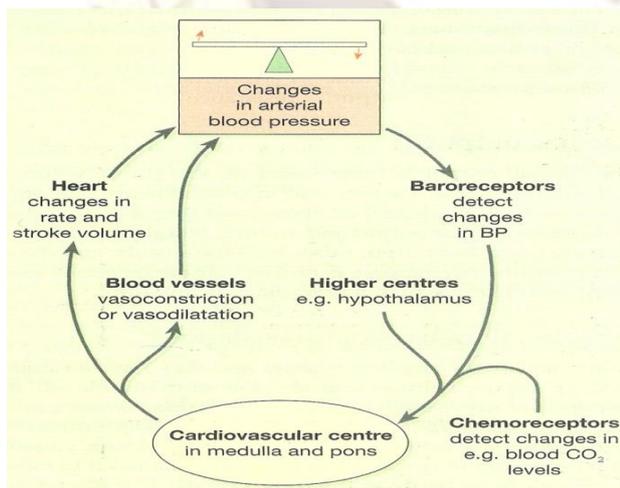


Figure 4: Summary of the main mechanisms in blood pressure control

3. Feedback systems (Loops)

A feedback system (loop) is a cycle of events, in which information about the status of a condition is continually monitored, and feedback to

a central control region. A feedback system consists of three basic components—control centre, receptor, and effector. Figure 5 shows the components (control centre, receptor, and effector) of a feedback system. The function of each component is explained below.

Control centre: The control centre determines the point at which some aspect of the body, called controlled condition should be maintained. In the body, there are hundreds of controlled conditions. A few examples are heart rate, blood pressure, and acidity of the blood, blood sugar level, body temperature, and breathing rate. The control centre receives information about the status of a controlled condition from a receptor and then determines an appropriate course of action.

Receptor: The receptor monitors changes in the controlled condition, and then sends the information, called the input, to the control centre. Any stress, that changes controlled condition is called stimulus.

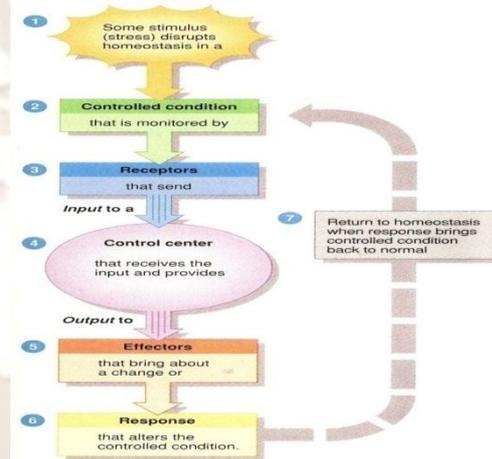


Figure 5: Components of a feedback system.

Effector:

The effector receives information, called the output, from the control centre and produces a response. The response that occurs is continually monitored by the receptor and feedback to the control centre, if the response reverses the original stimulus; the system is a negative feedback system. Negative feedback systems tend to maintain conditions that require frequent monitoring and adjustment within physiological limits.

Homeostasis of blood pressure:

Negative feedback

Blood pressure (BP) is the force exerted by blood, as it presses against the walls of the blood vessels, especially the arteries. When the heart beats faster or harder, BP increases; when blood volume increases, BP also rises.

If some stimulus (stress), either internal or external, causes blood pressure (controlled

condition) to rise, the following sequence of events occurs. The higher pressure is detected by pressure-sensitive nerve cells (the receptors) in the walls of certain arteries. They send nerve impulses (input) to the brain (control center), which interprets the impulses and responds by sending nerve impulses (output) to the heart (effector). Heart rate decreases and blood pressure drops (response). This returns blood pressure (controlled condition) to normal, and homeostasis is restored.

A second set of effectors also contributes to maintaining normal blood pressure. Small arteries, called arterioles, have muscular walls that can constrict or dilate upon receiving appropriate signals from the brain which is as shown in feedback loop in Figure 6.

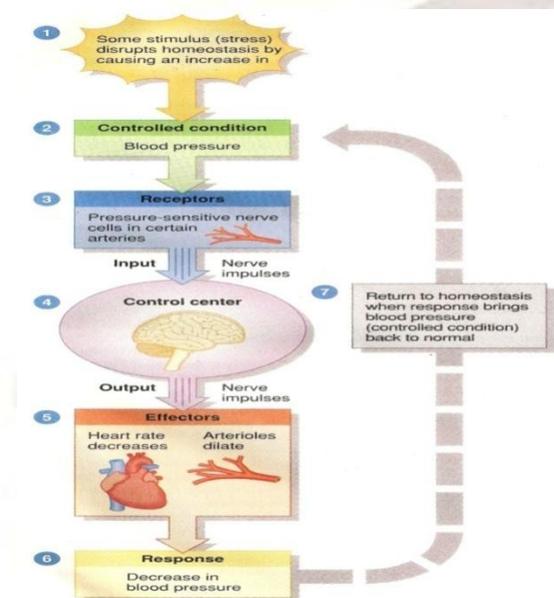


Figure 6: Negative feedback regulation of blood pressure via Baroreceptors

4. Complex Dynamics in Cardiovascular system

In Non-linear Analysis the regulation of blood pressure is analysed. The blood, heart, and blood vessels together make up the cardiovascular system [1,4].

The internal mechanism of cardiovascular system has two feedback loops.

1. First loop represents the effect of the baroreflex on heart rate.
2. Second loop represents the effect of the baroreflex on cardiac contractility, that effects stroke volume [6-10].

5. Cardio vascular center and regulation of arterial blood pressure

For a given cardiac output, circulatory mechanics determines the corresponding level of the arterial blood pressure. An electrical circuit

shown in figure 8 characterizes the circulatory mechanics. It consists of the aortic characteristic impedance (r), placed in series with a parallel combination of the peripheral resistance (R) and the total arterial compliance (C). The differential equation that describes the electrical circuit is given by

$$RC \frac{dP}{dt} + P = r RC \frac{dQ}{dt} + (R + r) Q$$

Where

P - Arterial blood pressure

Q - Cardiac output

C - Compliance

R - Peripheral resistance

r - Characteristic impedance

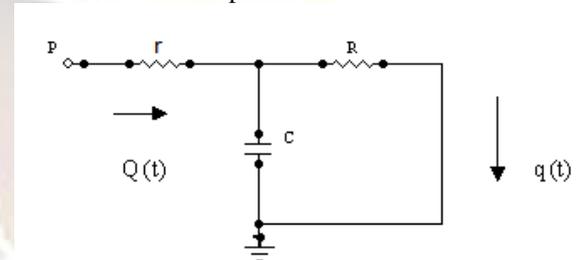


Figure 7: Electrical analog of circulatory Mechanism.

This circuit provides an output pressure that varies in proportion to cardiac period T .

$$Q = V_s / T$$

Now, we introducing time delay ($t-T$)

$$Q = V_s \times \frac{1}{T(t-\tau)} \text{ ----- (1)}$$

Where Cardiac period = $T(t-\tau)$

Effect of baroreflex on heart rate

The baroreceptors are used to sense the changes in pressure, and send this information back to the vasomotor center in the brain stem. The vasomotor center responds with changes in vagal and sympathetic nerve activity, which in turn modulates the cardiac period, T .

The dependence of cardiac period on pressure is given by

$$T(P) = T_{\min} + \frac{T_{\max} - T_{\min}}{1 + \gamma e^{-\alpha P / P_e}} \text{ ---- (2)}$$

Where T_{\max} and T_{\min} are the highest and lowest possible values for cardiac period T .

α and γ are the constants P_e – equilibrium level of arterial blood pressure. A schematic block diagram representing complete version of the model is displayed in Figure 8.

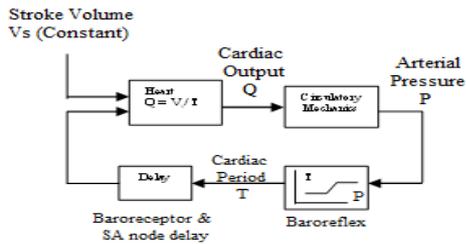


Figure 8: Cardiovascular variability model with single feedback loop

However, an overall delay τ is incorporated. This delay represents the combined lag associated with the baroreceptor response time and the response times of sinoatrial node to vagal and sympathetic stimulation. Using the simulink model of Figure 9 the changes in heart rate, with respect to time, and changes in cardiac output, with respect to blood pressure are obtained for different time delays.

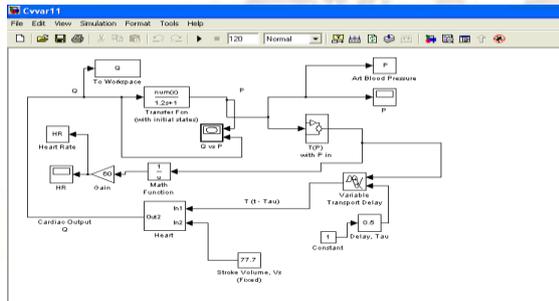


Figure 9: Simulink model implementation of the cardiovascular variability model with single feedback loop.

Heart block

It is having 2 inputs namely stroke volume V_s , which is a fixed value of 77.7 and the other input is time delayed cardiac period T ($t-\tau$). It provides an output (Q) that is given by equation (2) and is fed to the next stage. The internal diagram of this block is shown in Figure 10[5].

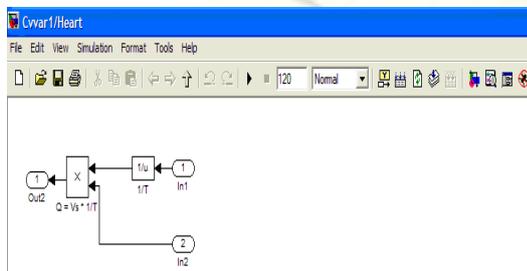


Figure 10: Internal diagram of the heart block

Transfer function block: The circulatory mechanism is characterized by an electrical circuit, shown in Figure 8, determines the level of arterial blood pressure for the given cardiac output. The

transfer function derived from the electrical circuit is given by

$$P / Q = \frac{0.468s + 0.93}{1.2s + 1} \text{----- (3)}$$

The output of this block is fed to the $T(p)$ block. **T (P) Block:** It gives the relation between cardiac period T and pressure P . It gives cardiac period as the output for the given input pressure by the relation given in equation (1) and is fed to the next stages. The internal block diagram of this block is shown in Figure 11.

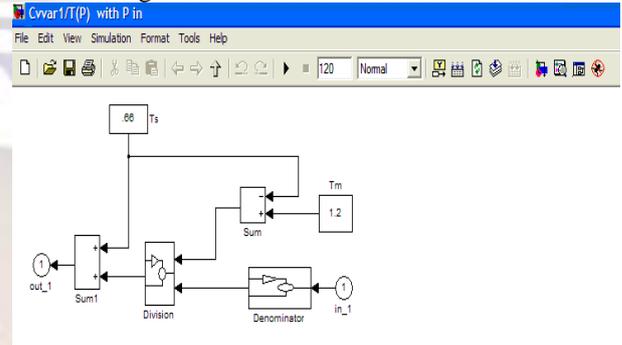


Figure 12: Internal diagram of the T (P) block

Changes in Pressure (P) are sensed by the baro receptors. Which relay this information back to the vasomotor centre in the brain stem. The vasomotor center responds with changes in vagal and sympathetic nerve activity, which in turn modulates the cardiac period T .

Table 2: Parameter values to have the steady state characteristics of the cardiovascular system

Sl. No.	Parameter	Value
1.	T_{Min}	0.66 Sec
2.	T_{Max}	1.2 Sec
3.	P_e	89 mm Hg
4.	α	31
5.	γ	6.7×10^{13}

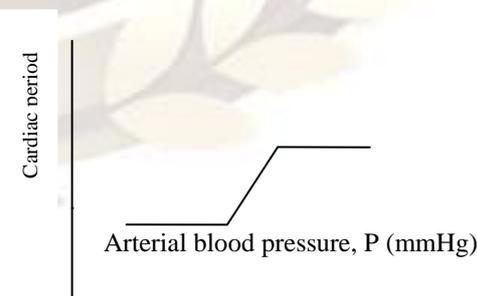


Figure 12: Steady state characteristics of the baro reflex

We assume stroke volume (V_s) to be independent of P . The steady state characteristics of this baro reflex response is shown in Figure 12, As P decreases, meaning that cardiac output increases,

since $Q = V_s/T$ and V_s is constant. Conversely, as P increases T also increases, decreasing Q . This accounts for the negative feedback effect of the baro reflex.

Math function: It is having an input of cardiac period T , and is converted into $1/T$, that refers to heart rate.

Gain: The amplitude of heart rate is multiplied by the value of gain and is fed to scope and to workspace. Here, the gain is chosen as 60.

Variable Transport delay: This block provides a time delay T ($t-\tau$) to the cardiac period, where t is taken as 3 sec and time delay (τ) is a variable value that depends on requirement, and this time delayed cardiac period is fed to heart block.

Scope: The scope is used to display the output. Here two scope blocks are used, first one is used to display the changes in arterial blood pressure and second scope is to display the heart rate.

Constant block: These blocks are used to provide a constant value. Here two constant blocks are used, one is to the delay block and the other is taken as stroke volume of 77.7 to the Heart. Thus the changes in arterial blood pressure cause changes in cardiac period, which in turn changes cardiac output.

Cardio vascular system with two feedback loops:

Dependence of V_s on P , which is also basically sigmoid in form as in the relation between T and P . Above 90 mm Hg, V_s remains relatively constant. Below 80 mm Hg, V_s decreases steeply with decreases in P . This is due to the concomitant increase in heart rate, which reduces the time of ventricular filling. As P decreases even further, V_s decreases to ward zero, as the heart begins to fail. Incorporation of the dependence of V_s on P produces a dual feedback-loop model. Furthermore, since cardiac output is the ratio of V_s to T , this introduces a nonlinear interaction between the two feedback loops. A schematic block diagram representing complete version of the model is displayed in Figure 13.

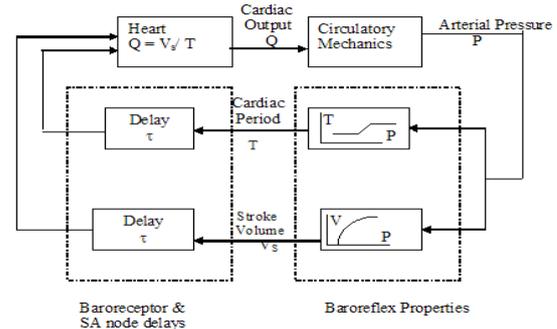


Figure 13: Cardiovascular variability and model with double feedback loop.

The model contains two feedback loops, one representing the effect of the baroreflex on heart rate, and the other representing the effect of the baroreflex on cardiac contractility, which in turn affects stroke volume.

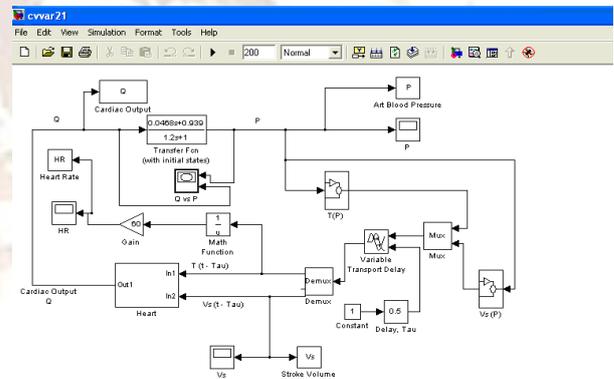


Figure 14: Simulink implementation of cardiovascular model with double feedback loops.

Simulink model of cardiovascular model with double feedback loops as shown in Figure 14 is used to obtain changes in heart rate with respect to time and changes in cardiac output with respect to blood pressure for different time delays.

The changes in heart rate are accomplished by an opposite change in stroke volume. Stroke volume can vary from a 70ml per stroke to about 200ml per stroke. The additional blocks used are given below.

Vs (P) block: This block refers to the variation in stroke volume with respect to changes in pressure. This is expressed as

$$V_s (P) = V_{\min} \frac{V_{\max} - V_{\min}}{1 + \gamma e^{-\alpha x P / P_e}} \text{ ---- (4)}$$

The output of this block is fed to scope and to the multiplexer. The internal diagram of $V_s (P)$ block is shown in Figure 15.

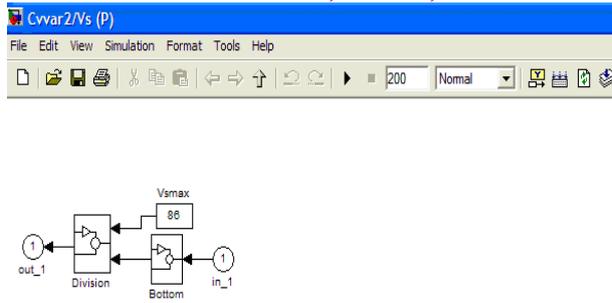


Figure 15: Internal diagram of Vs (P) block.

Multiplexer: This is having number of inputs and one output. It combines the output of Vs (p) block and T (P) block, and is fed to delay block.

6. Computation

- The values of aortic characteristic impedance(r), peripheral resistance(R), and compliance(C) are taken as $0.039 \text{ mm Hg S ml}^{-1}$, $1.9 \text{ mm Hg S ml}^{-1}$ and $1.333 \text{ ml mm Hg}^{-1}$, and these are substituted in the equation (2) to have the transfer function given by equation (3).
- The values shown in table2 are utilized in equation (3) to have the steady state characteristic of the cardiovascular system and is shown in Figure 12.
- The values of time delay are chosen as 0.5 sec, 1.2 sec, 1.8 sec and 2.5 sec and are substituted in figure 9 to 15 to have the time courses of heart rate and $x - y$ plots of cardiac output versus arterial blood pressure. These are shown in Tables 1 to 4.

7. Complex dynamics Analysis:

Table 1: Responses of the cardiovascular model at different time delays (Single Feedback Loop)

S.NO	Time delay- τ	Change in response	Simulation results
1	0.5sec	Time courses of heart rate having stright line	
2	1.2sec	System become oscillatory	
3	1.8sec	Periodic behaviour persists with increases in τ . The cycle duration of the oscillation, however increases as τ is increased.	
4	2.5sec	The cycle duration of the oscillation, however increases as τ is increased	

Table 2: Responses of the x-y plots of cardiac output at different time delays

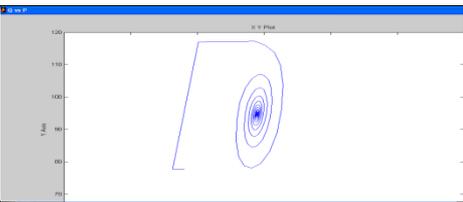
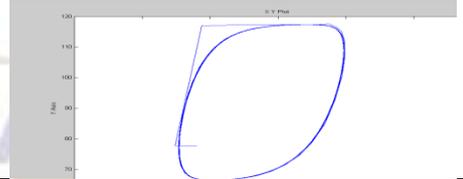
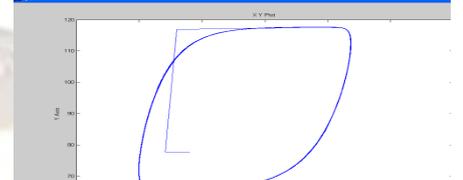
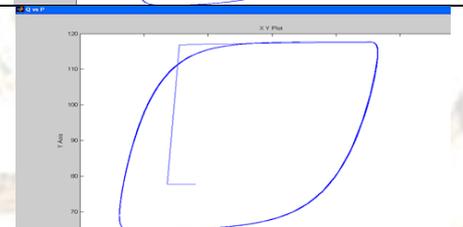
S.NO	Time delay- τ	Change in response	Simulation results
1	0.5sec	when τ is small ($\tau = 0.5\text{sec}$) the response rapidly converges to the equilibrium level.	
2	1.2sec	The prolongation of the time delays inherent in a closed loop system constitutes a highly destabilizing effect.	
3	1.8sec	The prolongation of the time delays inherent in a closed loop system constitutes a highly destabilizing effect.	
4	2.5sec	The prolongation of the time delays inherent in a closed loop system constitutes a highly destabilizing effect.	

Table 3: Responses of the cardiovascular model at different time delays (Two Feedback Loops)

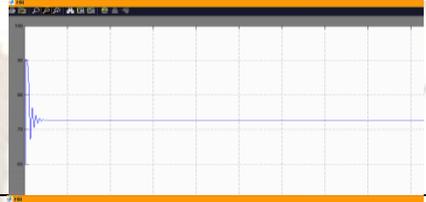
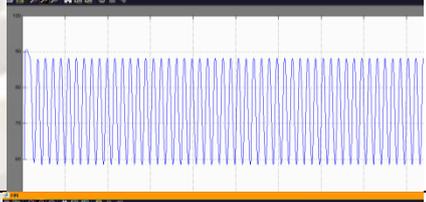
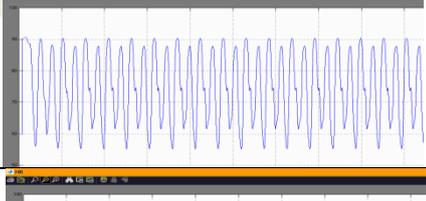
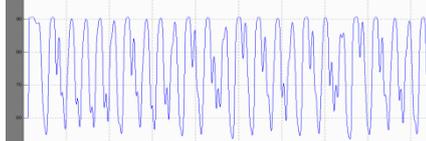
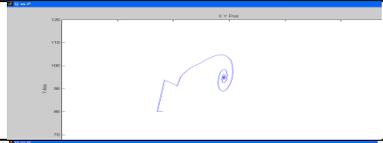
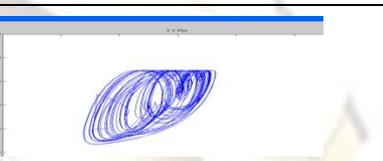
S.NO	Time delay- τ	Change in response	Simulation results
1	0.5sec	Time courses of heart rate.	
2	1.2sec	As τ is increased, a periodic oscillation develops as in the single feedback-loop case.	
3	1.8sec	The system remains periodic, however the oscillations now contain multiple frequencies, at a subharmonic and super harmonics.	
4	2.5sec	The oscillations turn into chaos.	

Table 4: Responses of the x-y plots of cardiac output at different time delays (Phase space plots, with Q plotted against P)

S.NO	Time delay- τ	Change in response	Simulation results
1	0.5sec	When time delay is small, the response rapidly converges to the equilibrium level, this is represented in the phase space.	
2	1.2sec	The phase space plots, with Q plotted against P	
3	1.8sec	The phase space plots, with Q plotted against P. Phase-space plot shows a complicated double loop.	
4	2.5sec	The phase space plots, with Q plotted against P.	

Conclusions:

- In the cardio vascular system, blood pressure is controlled by feedback loop through baro reflexes. In this system the changes in heart rate with respect to time and changes in cardiac output with respect to blood pressure are observed for different time delays.
- In the case of single feedback system, due to a small time delay of 0.5sec in cardiac period causes the response to converge rapidly to equilibrium point.
- If time delay is increased to 1.2sec, 1.8sec, and 2.5sec, then system becomes oscillatory and cycle duration increases with increase in delay.
- The system with double feedback loop exhibits the same response as single feedback at low frequencies. If time delay (τ) is increased, the system becomes periodic and periodic doubling occurs, and finally chaos results.

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